Clin. Cardiol. 20, 93–98 (1997)

Reviews

The Vital Role of Papillary Muscles in Mitral and Ventricular Function: Echocardiographic Insights

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Summary: The two left ventricular (LV) papillary muscles are small structures but are vital to mitral valve competence. Partial or complete rupture, complicating acute myocardial infarction, causes severe or even catastrophic mitral regurgitation, potentially correctable by surgery. Papillary muscle dysfunction is a controversial topic in that the role of the papillary muscle itself, in causing mitral regurgitation post infarction, has been seriously questioned; it is less confusing if this syndrome is attributed not only to papillary muscle but also to adjacent LV wall ischemia or infarction. Papillary muscle calcification is easily and frequently detected on echocardiography, but its clinical significance remains uncertain. Papillary muscle hypertrophy accompanies LV hypertrophy of varied etiology and may have a significant role in producing dynamic late-systolic intra-LV obstruction in hypertrophic cardiomyopathy and other hyperdynamic hypertrophied LV chambers. All the above abnormalities can be adequately assessed by 2-D echocardiography and the Doppler modalities. In selected cases, transesophageal echocardiography can provide additional valuable data by improving visualization of papillary muscles and mitral apparatus.

Key words: papillary muscle, left ventricular function, mitral regurgitation, echocardiography, hypertrophy, dysfunction

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Received: April 2, 1996
Accepted with revision: July 2, 1996

Introduction

Left ventricular (LV) papillary muscles are two small myocardial structures with an importance out of proportion to their size. The integrity of the mitral valve depends on the proper functioning of the papillary muscles. Two dimensional (2-D) transthoracic and transesophageal echocardiography (TTE and TEE) have made it possible to visualize papillary muscles and detect abnormalities of their structure and contraction, as well as of the underlying LV wall. Color flow Doppler has enabled the echocardiographer to assess the presence and severity of accompanying mitral regurgitation.

The spectrum of papillary muscle abnormalities ranges from a life-threatening emergency (papillary muscle rupture) to an echocardiographic finding of doubtful clinical significance (papillary muscle calcification). Some aspects of papillary muscles, such as simulation of mural ventricular thrombi, are of importance mainly to echocardiographers; other aspects such as the controversial and ambiguous entity of papillary muscle "dysfunction" are of wider clinical interest.

In this article we shall briefly review first the anatomy and then the echocardiography of papillary muscle abnormalities.

Anatomy (Fig. 1)

The left ventricle has two papillary muscles, the posteromedial and the anterolateral muscle; both arise from the LV free wall; no LV papillary muscle ever arises from the ventricular septum. Papillary muscles are conical, with a broad base that has myocardial continuity with the underlying LV wall. Trabeculations firmly fix the papillary muscle to the subjacent LV wall and to each other. Their bases are situated in the mid one-third of the LV chamber, but the apex or tip of the cone projects into the basal third of the LV. The anterolateral papillary muscle is slightly longer and narrower than the posteromedial muscle. The latter is more often split into two or more "heads." Both papillary muscles give chordae tendineae to both mitral leaflets: their location, just below the mitral commissures, is well suited for this purpose.

In uncommon cases, a third accessory papillary muscle is present, based closer to the LV apex than the normal muscles.

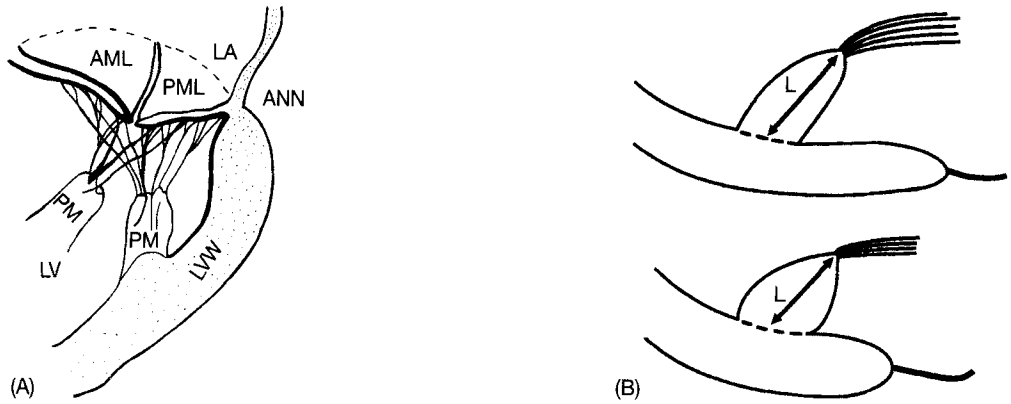

FIG. 1 (A) Diagrammatic section through LV to show normal papillary muscle anatomy. The papillary muscle (PM) shown arising from the posterior left ventricular wall (LVW) is the posteromedial one; the other PM is the anterolateral one. The anterior and posterior mitral leaflets (AML and PML) are attached to the mitral annulus (ANN and dotted line); both PMs have chordae tendineae attachments to both mitral leaflets. (B) Schematic representation of papillary muscle (PM) fractional shortening. Top: PM in end-diastole (A). Bottom: PM in end-systole (B). Fractional shortening: $\frac{A - B}{A} \times 100$. L = length.

The importance of such an extra papillary muscle near the apex is that it may be mistaken for mural thrombi, especially if that segment of LV happens to be infarcted.

The anterolateral papillary muscle has a dual blood supply, from the left anterior descending (LAD) coronary artery (second septal branch) and also usually from the circumflex coronary artery. The posteromedial papillary muscle gets its supply from the right coronary artery, although sometimes also from a circumflex branch. This muscle is said to be essentially dependent on one coronary artery and is therefore much more vulnerable to ischemic damage and rupture than the other (anterior) muscle. The tip or apex of the papillary muscle is more vulnerable to ischemia, which may be the reason why sclerosis or calcification of this apical region is frequently encountered as an incidental finding on 2-D echocardiography. It is believed that, in some individuals, the central artery of the papillary muscle is single and an "end artery" for practical purposes, whereas in others there are two or more arteries; the latter are less likely to suffer necrosis or sclerosis when the larger coronary arteries are narrowed or occluded by atherosclerosis.

Papillary Muscle Dysfunction

Papillary muscle length and function can be adequately assessed by either TTE or TEE.[1,2] Long-axis views of both the anterior and posterior papillary muscles can be obtained throughout the cardiac cycle by careful manipulation of the ultrasound beam during TTE or TEE. Fractional shortening of the papillary muscle can be determined as follows:

$$\frac{(\text{End-diastolic length} - \text{end-systolic length}) \times 100}{\text{End-diastolic length}}$$

Grimm *et al.*[3] studied papillary muscle function in dogs and found a mean percent fractional shortening of 22.8 ± 6.5 %. In normal humans, Kisanuki *et al.*[2] observed a mean percent fractional shortening of 30 ± 8%, while in patients with prior myocardial infarction, the mean percent fractional shortening was 15 ± 14%. Frequently associated with papillary muscle dysfunction is regional wall motion abnormality involving the posterobasal segment where the posteromedial papillary muscle frequently arises in such cases. Posteromedial papillary muscle motion is often reduced and appears more dense than the anterolateral papillary muscle. The short-axis views at the papillary muscle level, parasternal, and apical long-axis views are useful standard views in assessing papillary muscle morphology and function.

In a study of five patients with papillary muscle dysfunction and severe mitral regurgitation following acute myocardial infarction, Mintz *et al.*[4] observed that the posterior leaflet appeared to be retracted and held in a rigid position. Its motion was also noted to be reduced while the anterior leaflet motion was preserved. None of their patients had a flail leaflet, and systolic coaptation was normal in all patients.

More than 30 years ago, Burch *et al.*[5] proposed the concept of papillary muscle dysfunction as a possible etiologic mechanism for mitral regurgitation. Since then, papillary muscle dysfunction has been shown in several studies to be associated with varying degrees of mitral regurgitation.[5,6] This is often associated with a dilated and poorly contractile left ventricle and is an unlikely occurrence in the presence of good systolic performance.[2] Compared with normals, papillary muscles are significantly longer and thinner in dilated cardiomyopathy. In patients with reduced ejection fraction (EF) but normal LV chamber dimensions, no such differences are seen.[7]

Left ventricular dilatation, irrespective of etiology, has been commonly associated with papillary muscle dysfunction.[6] In fact, many years ago Shelburne *et al.*[8] postulated that in humans severe LV dysfunction must coexist with papillary muscle dysfunction to produce significant valvular insufficiency. Even in the setting of normal papillary muscle contractility, LV dilatation is thought to result in alteration of the spatial relationship between the papillary muscles, the chor-

dae tendineae, and the mitral valve orifice.[9] The valve leaflets are subsequently pulled further into the LV cavity, resulting in mitral valve incompetence and mitral regurgitation. In some situations, however, papillary muscle necrosis may result in acute fibrosis, shortening, and retraction of one of the mitral leaflets into the LV cavity, thus disrupting the usual line of anterior and posterior leaflet coaptation and leading to significant mitral regurgitation.[10]

The entire concept of ischemic mitral regurgitation resulting from papillary muscle dysfunction is currently undergoing considerable challenge. In fact, there is strong experimental evidence to suggest that isolated papillary muscle dysfunction in an adequately contractile ventricle is not associated with mitral regurgitation.[11–13] Recently, Kaul *et al.*[14] showed that ischemic mitral regurgitation was not related to either papillary muscle dysfunction or dysfunction of immediately adjacent myocardium but rather to global LV myocardial ischemia. In the study by Kisanuki *et al.*[2] using 2-D echocardiography, no significant association was found between mild mitral regurgitation and papillary muscle dysfunction. The occurrence of moderate to severe mitral regurgitation, however, was significantly more prevalent in patients with combined anterior and posterior papillary muscle dysfunction than in those with isolated papillary muscle dysfunction or normal papillary muscle function. Several interesting features were noted in that study: (1) Apparent mitral valve prolapse was noted only in a small number of patients with combined papillary muscle dysfunction. (2) While regional ischemia overlying the papillary muscles was frequently noted in patients with combined papillary muscle dysfunction, apparent incomplete mitral leaflet closure was not observed in any patients with combined papillary muscle dysfunction. (3) Several patients with combined papillary muscle dysfunction had no significant mitral regurgitation.

These findings suggest that papillary muscle dysfunction by itself may not be enough to cause significant mitral regurgitation. It is probable that an interplay of factors including global/regional LV systolic performance, LV cavity dimension, mitral leaflet morphology and function, as well as papillary muscle function play a role in the genesis of mitral regurgitation seen in patients with papillary muscle dysfunction. In an animal model, however, Kono *et al.*[15] have shown that ischemia of myocardial segments overlying the papillary muscles may be sufficient to produce mitral regurgitation.

The acute development of irreversible papillary muscle dysfunction without rupture and severe mitral regurgitation is an uncommon type of papillary muscle dysfunction.

Papillary Muscle Calcification (Figs. 2 and 3)

This condition was first diagnosed radiographically in 1974; after that, several isolated case reports appeared in

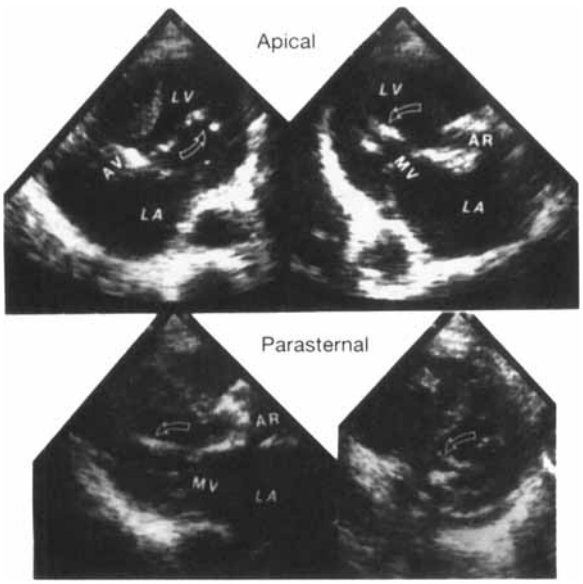

FIG. 2 Two-dimensional echocardiogram in apical 4-chamber view (top left), apical long-axis view (top right), parasternal long-axis view (bottom left), and parasternal short-axis view (bottom right). Arrows indicate papillary muscle calcification; two "heads" of the posteromedial papillary muscle are calcified. LV = left ventricle, LA = left atrium, AV = aortic valve, AR = aortic root, MV = mitral valve.

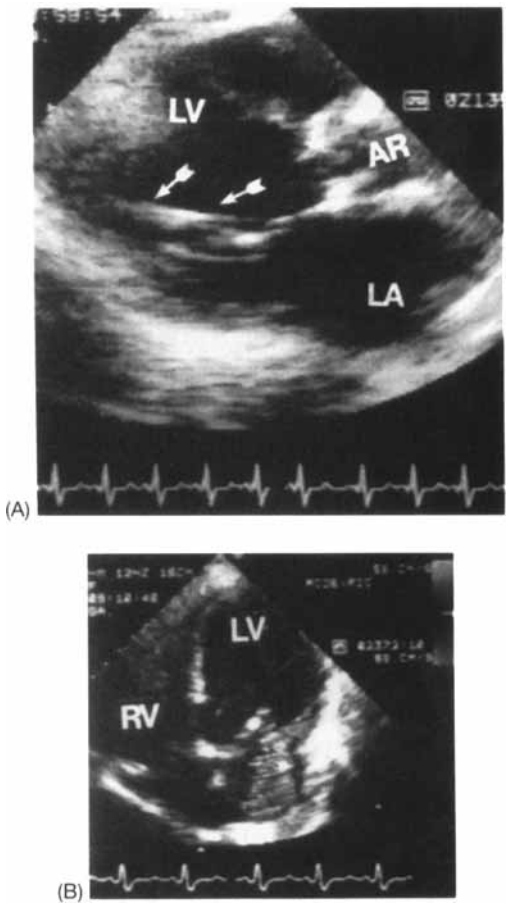

FIG. 3 (A) Parasternal long-axis view (below) showing extensive papillary muscle calcification (arrows). Some calcification is also present in the aortic valve. (B) Mitral regurgitant jet in the same patient.

which papillary muscle calcification (PMC) was visualized on 2-D echocardiography. Come and Riley[16] reported a series of 17 patients with PMC, in 5 of whom there was anatomic evidence of calcification in this location at surgery for mitral valve replacement. PMC is a common finding on routine echocardiography of elderly patients. The morphology and size of the calcification are very variable. PMC localized at the apex of the muscle is most frequent; sometimes the calcification has a cap or arrowhead shape occupying the narrow summit of the muscle cone. Occasionally, the PMC is heavy or extensive, extending through the whole length of the muscle from apex to base, even perhaps extending along mitral chordae tendineae.

Etiology

Coronary artery disease is often present and dilated cardiomyopathy is not an uncommon association. Several authors have reported some cases with myxomatous mitral valve changes. PMC is almost never encountered in young adults unless end-stage renal disease on long-term dialysis is present. Roberts and Cohen[9] called attention to LV calcification frequently occurring at three sites in elderly patients: aortic valve cusps, posterior mitral annulus, and papillary muscles. Every echocardiographer has seen many examples of such a triad (Roberts'). Other patients may exhibit only one or two of these three sites of calcifications.

Rarely does an inferior myocardial infarction or postinfarct aneurysm show excessive calcification on echocardiography and angiocardiogram. PMC may then constitute one component of the large myocardial calcific mass.

Papillary Muscle Rupture (Fig. 4)

Papillary muscle rupture is an uncommon life-threatening complication of acute myocardial infarction or trauma. It usually occurs 2 to 7 days post myocardial infarct and results in acute mitral regurgitation, pulmonary edema, and/or cardiogenic shock.[17] Without surgical intervention, mortality is estimated at 50% in the first 24 h and could reach 90% by 1 week. Wei *et al.*[18] estimated that 0.4 to 5.0% of all deaths after acute myocardial infarction are associated with papillary muscle rupture.

Papillary muscle rupture (like ventricular free wall rupture) is often associated with a first acute myocardial infarction[19] and can occur irrespective of infarct size. In fact, relatively small infarcts involving myocardial regions adjacent to the papillary muscle have been shown to lead to papillary muscle rupture.[18,20] The posteromedial papillary muscle, which often has a solitary source of blood supply from the posterior descending coronary artery, is thought to be more frequently involved, usually due to inferolateral myocardial infarction. The anterolateral papillary muscle receives its blood supply from two sources, the diagonal branch of the LAD and the left circumflex coronary artery, and is thus less frequently involved in papillary muscle rupture secondary to acute myocardial infarction. In fact, posteromedial papillary muscle rupture is three to six times more common than anterolateral papillary muscle rupture.[17–20] Unlike rupture of one of the muscle heads supporting the chordae tendineae, which often leads to acute mitral regurgitation or pulmonary edema, rupture involving the entire papillary trunk is rapidly and uniformly fatal.

When clinical findings are suggestive of papillary muscle rupture, echocardiographic information can be extremely useful in (1) making diagnosis of papillary muscle rupture, (2) excluding other mechanical complications of acute myocardial infarction such as ventricular septal rupture, (3) quantifying mitral regurgitation, and (4) evaluating LV function.

Two-dimensional TTE can be used for direct visualization of the papillary muscle rupture with associated flail mitral leaflet.[21] The parasternal and apical long-axis views appear to be most useful in assessing mitral valve function. Coarse diastolic fluttering of the flail leaflet may be seen associated with loss of systolic leaflet coaptation and profound systolic prolapse into the left atrium.[4] The anterior leaflet segment involved in the flailing motion is often long and redundant. Occasionally, the ruptured portion of the papillary muscle may appear as a mobile echogenic mass attached to the chordae tendineae and prolapsing into the left atrium.[4,21,22] This often has to be differentiated from a valvular vegetation, tumor or thrombus.

In some patients, adequate acoustic windows may not be easily obtainable by TTE. This is particularly so in patients in distress who are often tachypneic or those requiring mechanical ventilatory support. In these patients, TEE can be safely and rapidly performed at the bedside, even in critically ill patients, and will accurately demonstrate papillary muscle rupture as well as other associated problems.[23,24] The absence of acoustic interference results in superior visualization of intracardiac structures, including the left atrium and mitral apparatus.

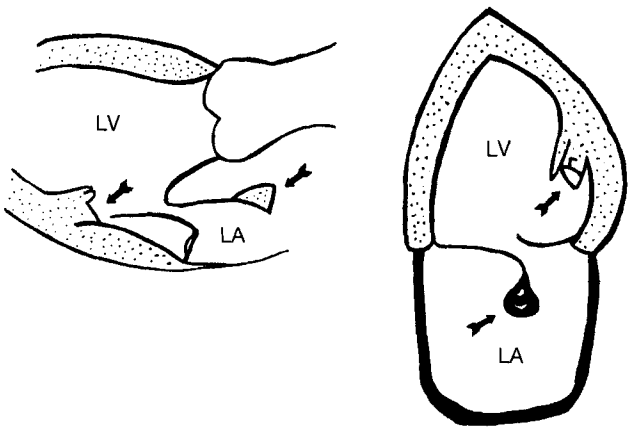

FIG. 4 Diagram showing parasternal long-axis view (A) and apical 4-chamber view (B) in a patient with papillary muscle rupture. In systole, the flail anterior mitral leaflet (with attached papillary muscle head) moves abnormally into the left atrium (LA) in systole, so that the mitral leaflets cannot coapt properly. Arrows indicate the stump of the ruptured papillary muscle.

Doppler interrogation greatly augments 2-D echo findings and has significantly improved the accuracy of echocardiographic diagnosis of papillary muscle rupture and associated mitral regurgitation.[22] Severe mitral regurgitation is readily identified by Doppler interrogation of a sample volume in the left atrium in the parasternal and apical long-axis views. Doppler color flow mapping has also proven very useful in the differentiation of papillary muscle rupture and ventricular septal defect when both are coexistent following acute myocardial infarction.[25] Using Doppler color flow mapping, Smyllie *et al.*[25] accurately identified mitral regurgitation in seven patients with isolated papillary muscle rupture. Doppler echocardiography was also particularly useful in localizing mitral leaflet abnormality by accurate determination of the direction of the regurgitant jet.

A flail posterior leaflet often results in an eccentric jet that is anteriorly directed to the interatrial septum; on the other hand, a flail anterior leaflet is associated with an eccentric jet directed to the posterolateral wall of the left atrium. Involvement of both mitral valve leaflets results in a centrally directed regurgitant jet.

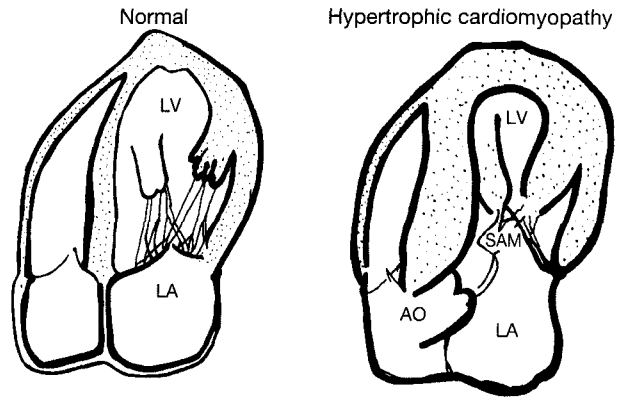

FIG. 5 Diagram of apical 4-chamber view, comparing normal appearances (left) with hypertrophic cardiomyopathy (right). In the latter, the LV chamber is smaller and there is conspicuous hypertrophy of the ventricular septum and papillary muscles. The origins of the papillary muscles appear displaced toward the LV apex. Abnormal systolic anterior mitral motion (SAM) is seen. LV = left ventricle, LA = left atrium, AO = aorta.

Papillary Muscle Hemorrhage

A recent report has directed attention to papillary muscle hemorrhage secondary to myocardial infarction as a cause of acute mitral regurgitation.[26] Using TEE, a marked and irregular echolucency of the posteromedial papillary muscle was visualized at the mid papillary muscle short-axis view. The systolic thickening of the papillary muscle as well as adjacent LV wall was impaired and appeared to prevent proper coaptation of the mitral valve leaflets. Diagnosis of papillary muscle hemorrhage was confirmed at surgery. The echocardiographic characteristics of papillary muscle hemorrhage as presented in this report appear consistent with the features described earlier by Hauser *et al.*[27] involving a hemorrhagic cyst of the papillary muscle.

The therapeutic approach to papillary muscle hemorrhage is similar to that of papillary muscle rupture. While temporizing measures with intra-aortic balloon pump and vasodilators may be initially helpful, the only definitive and effective treatment is surgical intervention.

Papillary Muscle Hypertrophy

The papillary muscles participate in hypertrophy of the LV myocardium due to hypertension, aortic stenosis, hypertrophic cardiomyopathy, end-stage renal diseases, and so forth. The abnormally thick papillary muscles in such cases may appear unduly large and conspicuous on 2-D echo, all the more so because the LV chamber is usually smaller than usual. Moreover, the papillary muscles may be in a somewhat abnormal LV location in hypertrophic cardiomyopathy; the protuberant ventricular septum and massive papillary muscles might so narrow the LV chambers as to cause intraventricular obstruction in some cases (Fig. 5). Madu *et al.*[1] have also recently shown that papillary muscle area systolic fractional shortening is reduced in the presence of LVH. In addition, while papillary muscle length in such hearts remains unchanged, its shape often changes (becoming thicker).

Left Ventricular Outflow Tract Obstruction

Few cases of left ventricular outflow tract (LVOT) obstruction due to papillary muscle abnormalities have been reported. This may result from hypertrophy, aberrant location, or abnormal attachment of the papillary muscle.[28–30] Clinical presentation and physical findings are often indistinguishable from those of aortic stenosis or hypertrophic obstructive cardiomyopathy. Marked mitral regurgitation may be present concomitantly due to loss of support for the anterior mitral valve leaflet. Secondary septal and LV free wall hypertrophy may be present. Two-dimensional and Doppler echocardiography represent the most reliable diagnostic tool both for anatomic and physiologic evaluation.

Echocardiography will reliably identify hypertrophied, aberrant, or abnormally attached papillary muscle. Doppler interrogation will document both the presence and severity of increased flow velocity and gradient in the LVOT as well as the severity and direction of the mitral regurgitant jet.

Conclusion

Left ventricular papillary muscles are vital to both left ventrilcular and mitral valve function. Papillary muscle rupture

and other abnormalities in papillary muscle morphology or contractile performance can be readily evaluated by 2-D and Doppler echocardiography.

TEE can be particularly useful in providing additional valuable information by dramatically improving image quailty of papillary muscles and mitral apparatus in selected cases.

The role of isolated papillary muscle dysfunction in causing mitral regurgitation is unclear and is the subject of ongoing debate.

Reference

1. Madu EC, Reddy RC, D'Cruz IA: TEE assessment of papillary muscle anatomy and contraction in patients with and without left ventricular hypertrophy. *J Am Coll Cardiol* 1996;27, No.2(suppl A):350A
2. Kisanuki A, Otsuji Y, Kuroiwa R, Murayama T, Matsushita R, Shibata K, Yutsudo T, Nakao S, Nomoto K, Tomari T, Tanaka H: Two-dimensional echocardiographic assessment of papillary muscle contractility in patients with prior myocardial infarction. *J Am Coll Cardiol* 1993;21:932–938
3. Grimm AF, Lendrum BL, Hum-Lin L: Papillary muscle shortening in the intact dog: A cineradiographic study of tranquilized dogs in the upright position. *Circ Res* 1975;36:49–57
4. Mintz GS, Victor MF, Kotler MN, Parry WR, Segal BL: Two-dimensional echocardiographic identification of surgically correctable complications of acute myocardial infarction. *Circulation* 1981;64:91–96
5. Burch GE, De Pasquale NP, Phillips JH: Clinical manifestations of papillary muscle dysfunction. *Arch Intern Med* (Chicago) 1963; 112–117
6. Phillips JH, Burch GE, De Pasquala NP: The syndrome of papillary muscle dysfunction, its clinical recognition. *Ann Intern Med* 1963; 59:508–520
7. Madu EC, Reddy RC, D'Cruz IA: Papillary muscle morphology and function in LV systolic dysfunction and dilated cardiomyopathy: A transesophageal echocardiographic study (abstr). *J Am Soc Echocardiogr* 1996;9:371 A
8. Shelburne JC, Rubinstein D, Gorlin R: A reappraisal of papillary muscle dysfunction. Correlative clinical and angiographic study. *Am J Med* 1969;46:862–871
9. Roberts WC, Cohen LS: Left ventricular papillary muscles. *Circulation* 1972;46:138–153
10. Saunders CA, Armstrong PW, Willerson JT, Dinsmore RE: Etiology and differential diagnosis of acute mitral regurgitation. *Prog Cardiovasc Dis* 1971;14:129–152
11. Miller GE, Kohn KE, Kerth WJ, Selzer A, Gerbode F: Experimental papillary muscle infarction. *J Thorac Cardiovasc Surg* 1968;56:611–616
12. Tsakiris AG, Rastelli GC, Amorim DD, Titus JL, Wood E: Effect of experimental papillary muscle damage on mitral valve closure in intact anesthetized dogs. *Mayo Clin Proc* 1970;45:275–285
13. Mittal AK, Langston M Jr, Cohn KE, Seltzer A, Kerth WJ: Combined papillary muscle and left ventricular wall dysfunction as a cause of mitral regurgitation: An experimental study. *Circulation* 1971;44:174–180
14. Kaul S, Sponitz WD, Glasheen WP, Touchstone DA: Mechanism of ischemic mitral regurgitation: An experimental evaluation. *Circulation* 1991;84:2167–2180
15. Kono T, Sabbah HN, Rosman H, Moshin A, Syed J, Stein PD, Goldstein S: Mechanism of functional mitral regurgitation during myocardial ischemia. *J Am Coll Cardiol* 1992;19:1101–1105
16. Come PC, Riley MF: M-mode and cross-sectional echocardiographic recognition of fibrosis and calcification of the mitral valve chordae and left ventricular papillary muscles. *Am J Cardiol* 1982; 49:461–466
17. Nishimura RA, Schaff HV, Shado C, Gersh BJ, Edwards WD, Tajik AJ: Papillary muscle rupture complicating acute myocardial infarction: Analysis of 17 patients. *Am J Cardiol* 1983;51:373–377
18. Wei JY, Hutchins GM, Bulkely BH: Papillary muscle rupture in fatal acute myocardial infarction: A potentially treatable form of cardiogenic shock. *Ann Intern Med* 1969;90:149–152
19. Barbour DJ, Roberts WC: Rupture of a left ventricular papillary muscle during acute myocardial infarction: Analysis of 22 necropsy patients. *J Am Coll Cardiol* 1986;8:558–565
20. Vladover Z, Edward JE: Rupture of ventricular septum or papillary muscle complicating myocardial infarction. *Circulation* 1977;55: 815–822
21. Buda AJ: The role of echocardiography in the evaluation of mechanical complications of acute myocardial infarction. *Circulation* 1991;84:I-109–I-121
22. Come PC, Riley MF, Weintraub R, Morgan JP, Nakao S: Echocardiographic detection of complete and partial papillary muscle rupture during acute myocardial infarction. *Am J Cardiol* 1985; 56:787–789
23. Sakai K, Nakamura K, Hosoda S: Transesophageal echocardiographic findings of papillary muscle rupture. *Am J Cardiol* 1991; 68:561–563
24. Manning WJ, Waksmonski CA, Boyle NG: Papillary muscle rupture complicating inferior myocardial infarction: Identification with transesophageal echocardiography. *Am Heart J* 1995;129:191–193
25. Smyllie JH, Sutherland GR, Genskens R, Dawkins K, Conway N, Roelandt JRTC: Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. *J Am Coll Cardiol* 1990;15:1449–1455
26. Cerza RF: Echocardiography of papillary muscle hemorrhage. *J Cardiothor Vasc Anesth* 1994;8(4):446–447
27. Hauser A, Rathod K, McGill J, Rosenberg BF, Gordon S, Timmis GC: Blood cysts of the papillary muscle. *Am J Cardiol* 1983;51: 612–613
28. Wada Y, Kawai T, Oga K, Oka T: Left ventricular outflow tract obstruction and mitral regurgitation caused by papillary muscle abnormalities: A case report. *J Thorac Cardiovasc Surg* 1993;106: 1223–1225
29. Wright PW, Witter RS: Obstruction of the left ventricular outflow tract by the mitral valve due to a muscle band. *J Thorac Cardiovasc Surg* 1983;85:938–940
30. Macartney FJ, Scott O, Ionescu MI, Deveral PB: Diagnosis and management of parachute mitral valve and supravalvar mitral ring. *Br Heart J* 1974;36:641–652